RESEARCH ARTICLE

The role of paternal obesity on the success of intracytoplasmic sperm injection cycle a tertiary IVF center in Turkey

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Abstract

Objective: To investigate the role of paternal obesity on intracytoplasmic sperm injection success.

Methods: The retrospective study was conducted in Suleyman Demirel University, Isparta, Turkey, from January 2015 to September 2017, and comprised data of infertile couples having undergone intracytoplasmic sperm injection cycle. The data was divided into three groups on the basis of paternal body mass index (BMI): normoweight NW (body mass index<23 kg/m^2), overweight OW (body mass index= 23-24.9 kg/m2), and obese group (body mass index ≥25 kg/m2). Fertilisation rate, count and quality of embryos, implantation, clinical pregnancy, take-home baby, abortion rates and sperm parameters were evaluated. SPSS 20 was used for data analysis.

Results: Of the 374 cases, 45(12%) were in NW group, 78(21%) in OW, and 251(67%) in obese group. The overall mean age of males was 34.60±5.80 years, and mean body mass index was 26.84±3.57 kg/m^2. There were no statistically significant differences in terms of fertilization rate, embryo count and quality, implantation, clinical pregnancy, take-home baby and abortion rates among the groups (p>0.05). Paternal obesity was not associated with sperm count and motility (p>0.05) either. Regression analysis showed that paternal obesity had no predictive effect on intracytoplasmic sperm injection success (p>0.05).

Conclusion: Excess weight in male partner had no effect on intracytoplasmic sperm injection success.

Keywords: Paternalinfluence, BMI, Intracytoplasmic sperm injection. (JPMA 69: 640; 2019)

Introduction

In vitro fertilisation (IVF) comprises 1.7-4% of all pregnancies with an increasing number due to the increasing prevalence of infertility.[1] It is an invasive and expensive treatment and also stressfull for the couples, therefore, it is important to seek approaches to increase IVF success.

Obesity is a public health problem which is commonly observed among both genders. Although its prevalence varies according to demographic features, it has been thought to be seen approximately 5% in developing countries and >50% in the developed ones.[2] Obesity and related healthy problems are increasing globally, especially in young population.[3] In addition to causing metabolic problems, it has negative impacts on reproductive system in both genders.[4] Several studies have focussed on the effect of maternal obesity on IVF outcome and it has been demonstrated that excess weight in females could deteriorate IVF success.[5] Although the prevalence of male infertility is increasing gradually,[6] there is limited information about the relation between paternal obesity and infertility and IVF outcome.

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It has been suggested that excess weight in male partner was an independent factor for infertility.[7] Obesity in males is more prevalent in the infertile population than the fertiles.[3] It has been also demonstrated that improving the metabolic health of obese males via exercise and/or diet could positively affect fertility status in both animals and humans.[8] Overweight and obese males are under risk of infertility due to the deleterious effect of weight on reproductive hormones.[9] It has been found that paternal body mass index (BMI) was correlated with decreased testesteron and increased oestrogen levels which resulted in hypogonadotropic hypoandrogenemia[10] via interfering with gonadotropin releasing hormone (GnRH) pulsatility[11] and this situation could cause compromised sperm production.[9] It has been demonstrated that increased paternal BMI is associated with decreased IVF success.[4,12] Some of the previous studies found that paternal overweight status was an independent negative factor for clinical pregnancy and live birth rates following IVF but not after intracytoplasmic sperm injection (ICSI) cycle.[13] In contrast, the others demonstrated no relation between increased paternal BMI and decreased clinical pregnancy rates after IVF.[14] Recent studies supported that paternal excess weight did not effect fecundity after IVF.[15]

As can be seen, the effect of paternal obesity on IVF success is controversial. Therefore, the current study was planned to assess the effect of paternal obesity on ICSI

success, including fertilisation rate (FR), transferrable embryo count (TEC), quality of embryo, implantation, clinical pregnancy (CP), take-home baby and abortion.

Materials and Methods

This retrospective study was conducted at the IVF unit of Suleyman Demirel University Faculty of Medicine, Isparta, Turkey, from January 2015 to September 2017. After approval was obtained from the institutional ethics committee, data was obtained from the hospital files of the patients undergoing ICSI cycle.

Age, weight, height, smoking, alcohol use, and the same characteristics of male partner, hormone levels on day 3 of cycle, thyroid stimulant hormone (TSH) and prolactin levels on a random day, aetiology of infertility, duration of infertility, previous ICSI/IVF cycle count (if performed) and intrauterine insemination cycle count, if performed, were recorded. Spermiogram parameters performed prior to treatment were also noted. Sperm samples were obtained via masturbation after abstinence of 3-5 days and evaluated according to World Health Organisation (WHO) criteria.[16] BMI of couples was calculated as weight (kg) divided by the height (m^2) squared. Normoweight NW group (BMI <23kg/m^2), overweight OW group (BMI= 23-24.99 kg/m^2), and obese group (BMI $\geq$25 kg/m^2).[17]

Data of patients with chronic diseases such as diabetes, and cardiovascular disease, endocrinopathies such as hyper/hypoprolactinemia and hypo/hyperthyroidism, hystory of malignancy, autoimmune disorders, chronic medication use, use of cocaine/opiates, and having a major andrologic problem and a history of vasectomy in the male partner was excluded. Frozen-thawed cycles were also excluded to rule out the detrimental effects of cryopreservation on the sperm deoxyribonucleic acid (DNA) via oxidative stress (OS).[18]

Recombinant follicle stimulating hormone (r-FSH) and/or urinary FSH (u-FSH) were administered for controlled ovarian stimulation (COS) on day 2 of cycle according to the female's age, ovarian grade, and BMI. GnRH antagonist was started when the follicle diameter was 13mm for down-regulation of pituitary gland. Cycle was monitored by serial transvaginal ultrasound (TV-USG) and blood samples (estradiol). Recombinant human chorionicgonadotrophin (r-hCG) was administered for triggering ovulation when at least 2 follicles reached 17mm in diameter. Then 36 hours after hCG administration, oocyte retrieval was performed by 17-gaugeneedle. Nuclear maturity of oocytes was evaluated under light microscopy and oocytes were classified as germinal vesicle, metaphase I, metaphase II (MII), degenerated, empty zona, or oocyte with anomaly. We defined MII rate as the number of MII oocytes divided by the number of total oocyte retrieved. ICSI procedure was performed on MII oocytes. Fertilisation was accepted as the presence of two pronuclei.

Quality of embryos were assessed under invert microscopy on day 3, when at least 8-cell stage, according to their morphological characteristics and classified from best quality Grade 1 to poor quality Grade 3. Embryos with even-sized blastomeres and/or <5% fragments were classified as Grade 1, embryos with slightly-moderate size differences in blastomeres and/or 5-50% fragments were classified as moderate quality Grade 2 and embryos with markedly different-sized blastomeres and/or >50% fragments were classified as Grade 3.

Embryo transfer at least 8-cell stage on day 3 was performed with respect to quality, cycle count and female age. Daily vaginal progesterone was performed for luteal support.

Fertilisation rate (FR) was calculated as the number of fertilised oocytes divided by the number of MII oocytes. Implantation was defined as an elevation in beta hCG (β-hCG) levels after 14 days of embryo transfer. CP was defined as the presence of an intrauterine gestational sac in TV-USG examination. Take-home-baby rate was defined as the birth of a neonate or after 24 weeks gestation. Abortion was defined as the termination of gestation before 20 weeks or birth of a foetus weighing less than 500 gram.

Primary outcomes were implantation, CP, take-home-baby and abortion rates.

Secondary outcomes were FR, transferrable embryo number, embryo quality, and sperm count and motility.

Statistical analysis was performed using SPSS 20. $P<0.05$ was accepted as statistically significant. Kolmogorov-Smirnov test was used to determine the distribution of continuous variables. One-way analysis of variance (ANOVA) was used to compare continuous variables(such as paternal age, maternal age, paternal BMI, maternal BMI, duration of infertility, etc.) between groups and pair wise comparisons of the groups were performed with least significant difference (LSD) test. Continuous variables were presented as mean$\pm$ standard deviation (SD) or medians and interquartile ranges (IQR: 25-75%) on the basis of their distribution. Categorical variables such as maternal and paternal habits (smoking, alcohol use, aetiology of infertility, etc) were compared by chi-square test and were presented as frequencies and percentages. Spearman's or Pearson's rank correlation analysis was used to assess the association of nonparametric and

parametric continous variables respectively. Logistic regression analysis was used to determine the association between dependent and independent variables. Covariates were chosen using prior knowledge about the known predictors affecting assisted reproductive treatment (ART) outcome.

Results

Of the 374 cases, 45(12%) were in NW group, 78(21%) in OW, and 251(67%) in obese group. The overall mean age of males was 34.60±5.80 years, and mean body mass index was 26.84±3.57 kg/m^2.

Of all the couples, 15(4%) were diagnosed as secondary infertile. The mean age of female partners was 31.63±5.67 years. Paternal age, maternal age, duration of infertility, previous IVF cycle, if performed, and previous intrauterine insemination (IUI) treatment, if performed, and basal hormone levels on day 3 of the cycle were found to be homogenously distributed among the groups (p=0.1, p=0.2, p=0.1, p=0.1, p=0.2, and p=0.05, respectively). Maternal BMI was significantly increased in obese group compared to OW (p=0.02) and NW (p=0.04) groups. Of all the female partners, 178(47.6%) were OW and obese. Of the all couples 101(27%) were diagnosed as male factor,

Table-1: Demographic features and baseline characteristics of groups in relation to paternal body mass index (BMI).

	NW (n=45)	OW (n=78)	Obese (n=251)	Total (n=374)	p value
Paternal age (years)[a]	32.91±6.12	34.61±5.70	34.90±5.74	34.60±5.80	0.1
Paternal BMI (kg/m^2)[a]	21.56±1.49	24.33±1.26	28.57±2.85	26.84±3.57	<0.0001
Paternal obesity (n, %)[b]	45/374 (%12.03)	78/374 (%20.8)	251/374 (67.11%)	329/374 (87.965%)	
Maternal age (years)[a]	30.31±6.2	31.89±6.02	31.78±5.43	31.63±5.67	0.2
Maternal BMI (kg/m^2)[a]	24.50±4.47	24.51±4.78	26.18±4.69	25.63±4.74	0.006*
Maternal obesity (n, %)[b]	14/45 (31.1%)	27/78 (34.6%)	137/251 (54.6%)	178/374 (47.6%)	0.004
Infertility duration (years)[a]	5.71±3.94	6.01±3.47	6.79±4.67	6.49±4.37	0.1
Cycle count (if performed)[a]	1.46±0.78	1.47±0.75	1.57±0.97	1.54±0.91	0.5
IUI (if performed)[a]	1.02±1.03	1.26±1.30	1.33±1.12	1.28±1.15	0.2
Maternal hormone levels[a]					
FSH (mIU/ml)	8.61±5.24	10±8.76	8.20±3.88	8.62±5.452	0.06
LH (mIU/ml)	5.36±3.34	6.33±5.11	5.76±4.96	5.83±4.82	0.06
E2 (pg/ml)	77.92±100.11	53.63±51.53	56.21±58.47	58.28±63.84	0.08
PG	1.34±1.79	0.79±0.62	0.91±1.08	0.93±1.12	0.06
TSH	2.12±1.01	1.97±1.26	1.99±1.01	2±1.06	0.7
Prolactin	13.07±5.10	14.95±8.80	15.15±10.53	14.86±9.69	0.4
Etiology of infertility[b]					
Female factor	22/45 (48.9%)	33/78 (42.3%)	113/251 (45%)	168/374 (44.9%)	
Male factor	12/45 (26.7%)	25/788 (32.1%)	64/251 (25.5%)	101/374 (27%)	0.7
Unexplained	11/45 (24.4%)	20/78 (25.6%)	74/251 (29.5%)	105/374 (28.1%)	
Maternal habits[b]					
Smoking use (%)	4/45 (8.9%)	8/78 (10.3%)	24/251 (9.6%)	36/374 (9.6%)	0.9
Alcohol use (%)	0/45 (0%)	0/78 (0%)	1/251 (0.4%)	1/374 (0.3%)	0.7
Paternal habits[b]					
Smoking use (%)	19/45 (42.2%)	26/78 (33.3%)	85/251 (33.9%)	130/374 (34.8%)	0.5
Alcohol use (%)	1/45 (2.2%)	3/78 (3.8%)	6/251 (2.4%)	10/374 (2.7%)	0.7
Sperm parameters[a]					
Count (X10^6)	62.03±63.62	57.79±68.52	66.80±69.68	64.35±68.67	0.5
Motility (%)	41.40±26.91	42.56±24.13	44.04±24.90	43.41±24.94	0.7
A (%)	11.64±13.06	12.02±15.05	11.86±12.71	11.87±13.24	0.9
B (%)	28.57±21.44	30.69±18.96	31.82±20.39	31.19±20.20	0.5
C (%)	11.33±9.20	12.70±9.61	12.81±10.40	12.61±10.08	0.6

p values were presented among all groups.

a: One way analysis if variance (ANOVA).

*: p value is significant between normoweight and obese group (p=0.04) and between overweight and obese group (p=0.02) by LSD test. p value is not significant between normoweight and overweight gorup (p=0.8) by LSD test.

b: χ2-test

BMI: Body mass index; FSH: Follicle stimulating hormone; LH: Luteinizing hormone; E2: Estradiol; PG: Progesteron; TSH: Thyroid stimulant hormone; IUI: Intrauterine insemination; A: Percent of fast progressive motile sperm; B: Percent of slow progressive motile sperm; C: Percent of non-progressive sperm; OW: Overweight; NW: Normal weight; IUI: I ntrauterine insemination; LH: Luteinizing hormone; FSH: Follicle stimulating hormone; E2:Estradiol; PG: Prostagladin; TSH: Thyroid-stimulating hormone.

Table-2: Ovarian stimulation characteristics and oocyte retrieval parameters among groups.

	NW (n=45)	OW (n=78)	Obese (n=251)	Total (n=374)	p value
Gonadotropin type[b]					
r-FSH	13/45 (28.9%)	22/78 (28.2%)	74/251 (29.5%)	109/374 (29.1%)	0.9
r-FSH (+) u-FSH	32/45 (71.1%)	56/78 (71.8%)	177/251 (66.8%)	265/374 (70.9%)	
Starting dose of r-FSH[a]	258.33±65.06	246.79±59.53	240.28±61.55	243.81±61.69	0.1
Starting dose of u-FSH[a]	127.27±39.70	132.14±37.75	135.38±42.27	133.73±41.02	0.5
Total oocyte number[a]	8.08±5.99	7.87±6.75	9.24±7.04	8.81±6.87	0.2
MII oocyte number[a]	5.88±4.51	5.52±4.92	6.17±4.66	6±4.69	0.5
MII Rate (%)[a]	73.22±23.03	69.78±27.05	69.45±21.80	69.96±23.09	0.6
Transfer day[b]					
Day 3	21/35 (60%)	28/50 (56%)	103/186 (55.4%)	152/271 (56.1%)	
Day 4	4/35 (11.4%)	14/50 (28%)	43/86 (23.1%)	61/271 (22.5%)	0.3
Day 5	10/35 (28.6%)	8/50 (16%)	40/186 (21.5%)	58/271 (21.4%)	
Transferred embryo number[b]					
SET	27/35 (77.1%)	41/50 (82%)	142/187 (75.9%)	210/272 (77.2%)	0.6
DET	8/35 (22.9%)	9/50 (18%)	45/187 (24.1%)	62/272 (22.8%)	

a: One way ANOVA
b: χ2-test
r-FSH: Recombinant follicle stimulating hormone; u-FSH: Urinary follicle stimulating hormone; MII: Metaphase II; SET. Single embryo transfer; DET: Diembryo transfer.OW: Overweight; NW: Normal weight.

Table-3: The effect of paternal obesity on IVF outcome (primer outcome).

	NW (n=45)	OW (n=78)	Obese (n=251)	Total (n=374)	p value
Fertilization[b]	37/40 (92.5%)	54/66 (81.8%)	192/219 (87.7%)	283/325 (87.1%)	0.5
FR (%)[a]	58.56±29.47	48.71±35.49	54.63±31.74	53.91±32.31	0.2
Transferrable embryo number[a]	3.70±2.72	3.34±3.41	3.70±3.03	3.63±3.07	0.5
Embryo quality[b]					
Grade A	31/36 (86.1%)	48/51 (94.1%)	168/189 (88.9%)	247/276 (89.5%)	
Grade B	4/36 (11.1%)	2/51 (3.9%)	18/189 (9.5%)	24/276 (8.7%)	0.7
Grade C	1/36 (2.8%)	1/51 (2%)	3/189 (1.6%)	5/276 (1.8%)	
Implantation (n, %)[b]	4/33 (12.1%)	13/50 (26%)	37/188 (19.7%)	54/271 (19.9%)	0.3
Clinical pregnancy (n, %)[b]	3/33 (9.1%)	11/50 (22%)	32/188 (17%)	46/271 (17%)	0.3
Take home baby n, (%)[b]	3/33 (9.1%)	7/151 (14%)	30/188 (16%)	40/271 (14.8%)	0.5
Abortion (n, %)[b]	1/33 (3%)	7/50 (14%)	14/188 (7.4%)	22/271 (8.1%)	0.1

a: One way ANOVA
b: χ2-test
IVF: In vitr fertilisation
FR: Fertilization rate.
OW: Overweight
NW: Normal weight.

168(44.9%) as female factor and 105(28.1%) as unexplained infertility. The aetiological factors were found to be distributed homogenously among the groups (p= 0.7). Mean total sperm count was 64.35±68.67 million/mL and the percent of progressive sperm motility was 43.41%. Mean total sperm count was also found to be distributed homogenously among the groups (p=0.5, p=0.7, p=0.9, p=0.5, and p=0.6, respectively). Smoking and alcohol use were also comparable among the groups (p=0.05) (Table-1).

Both u-FSH and r-FSH were used for COS in 265(70.9%) patients. Gonadotropin type used for COS was similar in the three groups (p=0.9). GnRH antagonist was used for down-regulation of pituitary gland in all patients. Starting dose of r-FSH and u-FSH were similiarin the groups (p=0.1 and p=0.5, respectively). Oocyte retrieval was performed in all patients, but at least one MII oocyte was retrieved in 333(89%) couples. Response to ovarian stimulation parameters, including total retrieved oocyte number, MII oocyte, and MII rate, were similar across all categories of male BMI (p=0.2, p=0.5, and p=0.6, respectively). Single

embryo transfer was performed in 210(77.2%) of 272 cycles and diembryo transfer was performed in 62(22.8%) of 272 cycles. There was also no statistical difference in terms of transferred embryo number and transfer day among NW, OW and obese groups (p=0.6 and p=0.3, respectively). There were 271 cycles with an embryo transfer: 152(56.1%) were day 3 embryo transfers, 61(22.5%) were day 4, and 58(21.4%) were day 5 transfers (Table-2).

Of the 374 cycles initiated, 41(11%) had no MII oocytes retrieved and in 8(2%) cases, no sperm was obtained via testicular sperm extraction. The fertilization, as such, was assessed for the remaining 325(87%) cycles. Fertilisation and FR were not associated with paternal BMI (p=0.5 and p=0.2). Transferrable embryo number and embryo quality were also distributed homogenously among the groups (p=0.5 and p=0.7). Embryo transfer was performed in 271(72.4%) of 374 cycles. Overall, there were 54(19.9%) positive β-hCG levels, 46(17%) CPs and 40(14.8%) live births. Implantation, CP and take-home-baby rates were comparable among the groups (p=0.3, p=0.5, and p=0.1).ART was also similar in the groups (p=0.1) (Table-3).

Multivariate regression analysis was performed to evaluate the effect of paternal BMI on ICSI outcome. For fertilization after adjustment of maternal age, maternal obesity and aetiology of infertility, paternal obesity was not predictive for fertilisation. Only maternal age had negative predictive effect on fertilisation (β=-0.9, p=0.004, odds ratio [OR]; 95% confidence interval [CI]= 0.91 [0.58-0.97]). When same parameters and embryo transfer day, quality of embryo and transferred embryo number were taken as covariates, paternal obesity and none of the covariates were predictive for implantation, CP and take-home-baby rates (p>0.05).

In correlation analysis, paternal BMI was positively correlated with maternal BMI (r=0.17, p=0.001). Paternal age, duration of infertility, FR, transferrable embryo number, total sperm count, sperm motility, cycle count and IUI count were not correlated with paternal BMI (p>0.05 each).

Discussion

The study evaluated the effect of paternal obesity on IVF success via determining FR, embryo quality, transferrable embryo count, implantation, CP, take-home-baby and abortion rates and also sperm parameters in 374 couples undergoing ICSI cycle. These were adjusted for the most important female characteristics that are known to have critical effect on IVF outcomes.

It has been demonstrated that 52.7% of males older than 15 years are either OW or obese in Turkey.[19] That 87.96% of males in our study were obese and OW suggests a link between paternal obesity and infertility. Campell et al. also found increased infertility in obese males, which is consistent with our study.[20]

There was no significant relation between paternal obesity and FR, implantation, CP, take-home-baby rates, and abortion rates in the current study. In NW group, clinical success parameters of ICSI, including implantation, CP, and take-home-baby rates, were non-significantly decreased compared to OW and obese groups, but this could be due to the small sample size of NW group. Negative impacts of female obesity on IVF outcome have been demonstrated,[5] but there are limited studies related to the effect of paternal obesity on the success of IVF. No association was found between paternal BMI and FR,[12] but it has been shown that increased paternal BMI negatively affects both CP and live birth rates in ICSI cycles.[4,12] Colaci et al found no association between paternal obesity and FR, implantation, CP rate, and an inverse relation between men's BMI and live birth rate among couples undergoing ICSI cycle.[14] Umul et al. found that CP and live birth rates decreased with increased paternal BMI, but FR and implantation were not affected by paternal obesity.[4] In a rodent model, paternal obesity has shown negative effects on implantation.[21] Campell et al. found reduced live birth rate and pregnancy viability with an absolute risk of 10%, but non-significant reduction in CP rate between obese and NW males in IVF population by investigating 30 studies with a total of 115,158 couples in their meta analysis.[20] Consistent with our results, Keltz et al. could not find a relation between paternal obesity and pregnancy rates in ICSI cycles.[13] In a recent meta analysis of 10 cohort studies involving 5262 male partners, paternal excess weight had no negative effects on CP and live birth rates and also sperm parameters in both IVF and ICSI cycles.[22] The mechanism underlying the inability to link negative IVF results and increased BMI remains unclear. It has been suggested that ICSI could overcome the negative impact of obesity on oocyte-spermatozoon interaction.[13] Another reason related to the absence of association between paternal obesity and IVF outcome is that maternal characteristics, including age, BMI, basal hormone levels on day 3 of cycle, have more essential roles than paternal obesity on IVF outcome. Disparateness in the identification of clinical characteristics, including identification of CP, classification of weight status according to BMI, simple and adjusted outcomes, could also lead to different results among studies. Clinical pregnancy rates were reported as: heartbeat detected per oocyte retrieval,[12]

USG confirmation (giving no further details) per embryo transfer cycle,[14] intrauterine gestational sac on TV-USG per cycle,[13] heartbeat detected per ICSI cycle,[4] and gestational sac and/or clinical recording of heartbeat (or documentation of birth or termination) per cycle.[15] Definition of live birth rate was also different between studies: per oocyte retrieval,[12] embryo transfer,[14] treatment cycle, ICSI cycle,[4] and IVF cycle.[15] Lack of the evaluation of oocyte quality which is closely associated with IVF outcome in the studies could also explain the discrepancy in results. Non-significant but lower implantation, CP, and take-home-baby rates in NW group than OW and obese groups in our study could either be a chance finding or due to unmeasured confounding factors that could not be adjusted.

We also found that paternal obesity was not predictive of fertilisation after adjustment of maternal age, maternal obesity and aetiology of infertility; only maternal age had negative predictive effect on fertilisation. When the same parameters and embryo transfer day, quality of embryo and transferred embryo number were taken as covariates, none of the cofactors were predictive for implantation, CP and take-home-baby rates. Colaci et al. found paternal obesity had negative predictive effect on live birth rates.[14] The results are controversial because some of studies adjusted the data, but others did not. Unadjusted and cofactors, like age, day 3 FSH level, female BMI, aetiology of infertility, day of embryo transfer, stimulation protocol (agonist versus antagonist) and semen quality parameters were different in various studies.

No relation between transferrable embryo number, embryo quality and paternal obesity was demonstrated in our study. Consistent with our study, no association was found between proportion of poor quality embryos, early embryo development and paternal obesity.[14] Also, no differences have been found in terms of day 5 embryo quality between obese and NW males.[15] However, in a rodent model, paternal obesity has shown negative effects on embryo development and blastocyst viability.[21] A significant decrease was found in blastocyst development with increasing BMI, but there was no association with embryo grade on day 3.[12]

We found no association between paternal obesity and total sperm count and motility. The results of studies related to the effect of paternal obesity on sperm parameters are controversial. Some studies found no association between increased paternal BMI and poor sperm parameters,[23] but others showed that paternal excess weight could influence sperm motility, concentration and total count.[4,10] No differences in terms of clinical sperm parameters, assessed using WHO criteria, including concentration and progressive motility, were found among IVF couples in a meta analysis.[20] Discrepancy in the classification of participants according to BMI and also different methods for semen analysis could be the causes of different results. Excess weight in males can lead to oligospermia by causing decreased levels of sex hormone-binding globulin and total testosterone.[24] Spermatogenesis is under control of free testerone and FSH which could be slightly influenced by overweight and obesity.[25] Therefore, it is understandable that increased BMI could not affect sperm parameters in spite of an alteration in endocrinologic profile.

We also found a positive correlation between paternal BMI and maternal BMI, but paternal age, duration of infertility, FR, transferrable embryo number, total sperm count, sperm motility, cycle count and IUI count were not correlated with paternal BMI. In contrast to our study, Umul et al. demonstrated a correlation between paternal BMI and previous cycle count.[4]

Retrospective design and small sample size were the major limitations of our study. The lack of the assessment of sperm quality is another limitation. To reduce the limitations, multivariate regression analysis was performed and covariates were chosen using prior knowledge about the known factors affecting IVF outcome. Frozen cycles were excluded to rule out the negative impact of criopreservation on sperm DNA damage. It is not possible to generalise our results because the study population represented data related to couples undergoing ICSI treatment during the study period. Also, the results need to be confirmed by further studies with a large sample size.

Conclusion

There was no evidence of negative impact of paternal obesity on fertilisation, quality of embryo, transferrable embryo number, implantation, CP, take-home-baby rates, and total sperm count and motility among couples undergoing ICSI cycle.

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